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Normal intracardiac and great artery blood velocity measurements by pulsed Doppler echocardiography

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SUMMARY One hundred and 10 normal subjects were studied by pulsed Doppler velocimetry to determine the range of values of blood velocity across the cardiac valves and in the great vessels. Modal peak velocities of 1·55 m/s occurred in the left heart, but right heart peak velocities were lower. In most sites a statistically significant inverse relation between peak velocities and age or body surface area was found. Time to peak velcocity in the pulmonary artery and ascending aorta increased significantly with age and was shorter in the aorta than in the pulmonary artery. These data were developed to serve as standards for the assessment of values recorded in patients with congenital and acquired cardiac disease.

The Doppler shift of ultrasound can be used to determine blood velocity at a number of cardiac and vascular sites, and these data in turn may be used to calculate blood flows and pressure differences.[1–3] Several small series of patients with normal values have been reported. Hatle and Angelsen reported the results in 30 children and 40 adults, but no methodology was detailed.[4] Grenadier and colleagues reported values for a small group of children, but no effort was made to correct velocities for the elevational plane angle, and aortic velocities were not measured with currently available transducers which can record velocities parallel with blood flow in the ascending aorta.[5] Several other reports mention normal velocities or time to peak velocity at isolated sites.[6–8]

The purpose of this study was to determine the modal peak blood velocity and time to peak velocity for a normal population at several clinically important sites. A feature of this study is that all measurements were obtained after careful alignment of the ultrasound beam with flow in three planes.

Patients and methods

One hundred and 10 normal volunteers with no evidence of cardiac disease or other known chronic illness were studied. None had been referred for cardiac evaluation. Informed consent was obtained from each subject or from the parents of the younger children. All were studied at rest and without sedation by one of two examiners (NW or SJG). The ages of the subjects ranged from 14 days to 35 years (mean 11·6 years).

Each examination was performed with an instrument which combined range gated pulsed Doppler velocimetry and cross sectional imaging echocardiography (Honeywell Ultra Imager). Velocities were obtained by aligning the Doppler cursor with the expected direction of flow in the two visualised planes and then searching in these two planes for the signal with the highest velocity and least spectral dispersion. The transducer was then angled in the third, or elevational, plane to achieve the highest velocity with the least spectral dispersion.

The velocity thus obtained was considered to be the maximal velocity. This was measured from baseline (zero) velocity to the peak of the modal velocity, identified as the darkest point at the peak of the curve (Fig. 1a). This point was chosen in preference to the absolute maximum velocity seen at the peak for reasons which will be discussed. The mean of four or five complexes was recorded. Time to peak velocity was measured from the departure of the velocity curve from the baseline to the modal peak velocity. Modal peak velocity and time to peak velocity were recorded in the superior vena cava, the ascending aorta and descending aorta (from the suprasternal notch), the abdominal aorta (from the subxiphoid region), the inlet of the tricuspid and mitral valve (from the apical four chamber plane), and the main pulmonary artery

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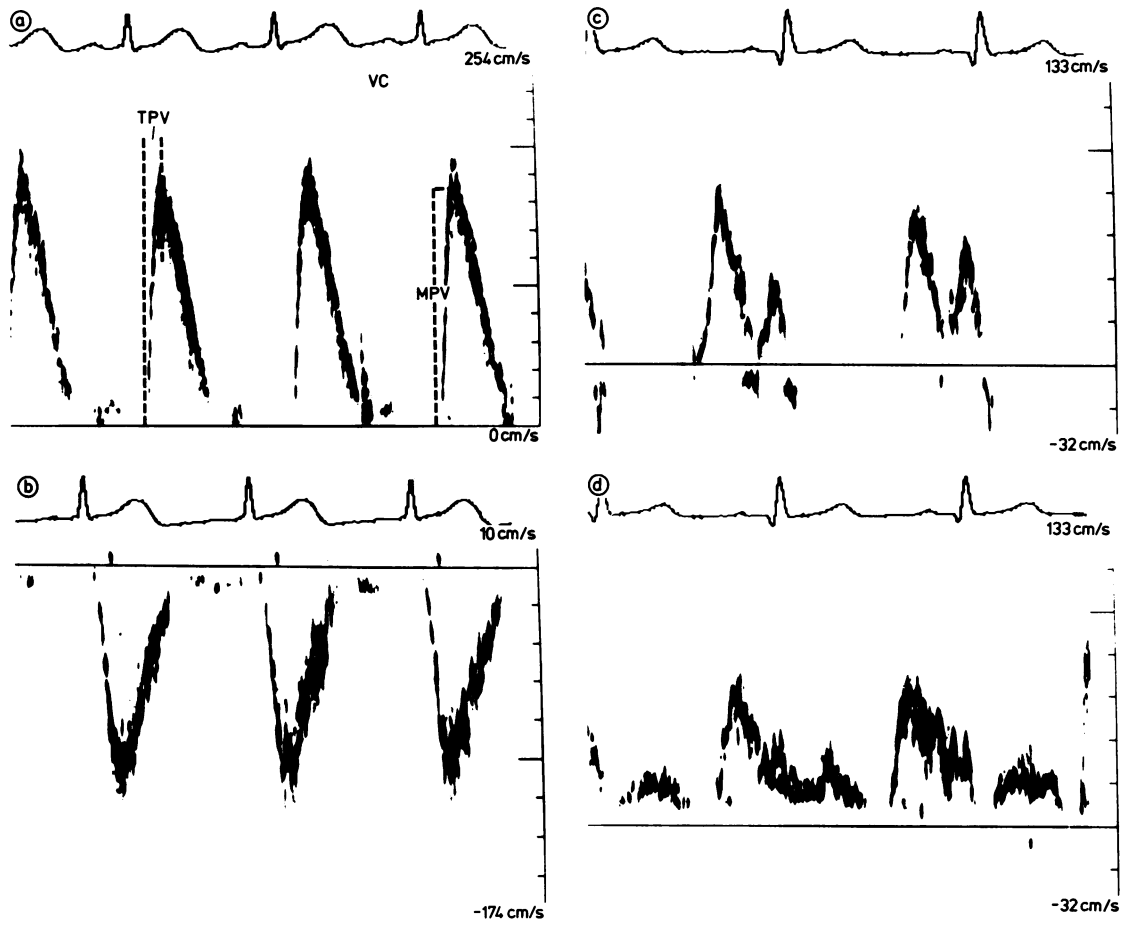

Fig. 1 *Pulsed Doppler echocardiograms showing a normal waveform (a) in the ascending aorta (viewed from the suprasternal notch), (b) in the pulmonary artery (parasternal short axis view); (c) across the mitral valve (apical four chamber view); and (d) across the tricuspid valve (apical four chamber view). (a) The Doppler frequency shift is converted to a velocity measurement in cm/s (vertical axis). A systolic waveform directed towards the transducer (positive wave) is present. The ascending limb of the wave is a discrete line indicating that at any given point all blood cells within the sample volume are travelling in the same direction at similar speed. At peak velocity there is some broadening of the line indicating blood cells travelling in differing directions and velocities. This feature is also apparent during the deceleration phase of the curve. The modal peak velocity (MPV) is the velocity that occurs most frequently and is identified as the darkest point on the peak of the curve. Time to peak velocity (TPV) is measured from the onset of the upstroke of the curve to the MPV as shown. VC, valve click. (b) A negatively directed systolic wave with characteristics similar to those in the aorta is present. (c) Two positively directed waves are present in diastole; the first and highest peak (from which modal peak velocity is measured) corresponds to the period of maximum mitral flow and the second coincides with the flow associated with atrial contraction. (d) The general configuration of the waveform is similar to that across the mitral valve but modal peak velocity is lower.*

immediately distal to the pulmonary valve (from the parasternal short axis view). Time to peak velocity and modal peak velocity were measured from the monitor at a sweep speed of 100 mm/s with electronic calipers. Subjects were not restudied by the other operator nor were measurements repeated at different times of the same day or on different days.

Although it was possible to record velocities from all other areas at an intercept angle of $\leq 20°$ in the two visualised planes, the abdominal aorta was always interrogated at an intercept angle of $>20°$. Therefore at this site velocities were corrected according to the

Table 1 *Modal peak velocity and time to peak velocity in the heart and great vessels in 110 subjects*

Site	*Range*	*Mean*	*Median*	*1 SD*
	Modal peak velocity (cm/s)			
Superior vena cava	28–80	51	51	13
Tricuspid valve	33–81	53	51	12
Pulmonary artery	52–131	81	82	17
Mitral valve	44–128	77	78	16
Ascending aorta	76–155	104	103	19
Descending aorta	70–160	101	99	17
Abdominal aorta	55–222	113	107	30
	Time to peak velocity (ms)			
Pulmonary artery	60–180	121	120	27
Ascending aorta	50–150	92	90	23
Descending aorta	50–190	107	110	28

formula: true velocity = recorded velocity/cosine theta, where theta is the angle at which the ultrasound beam intercepts the axis of blood flow.

STATISTICAL ANALYSIS

For each variable means and standard deviations were calculated. Scatter diagrams for all peak velocities and time to peak aortic and pulmonary velocities against age and body surface area were plotted and correlation coefficients and standard errors of the estimate determined assuming a linear relation. To facilitate statistical analysis for those subjects in whom complete data were not obtained the respective mean value of the peak velocity calculated from the remaining population for that variable was used. This method was not, however, used to complete data for the abdominal aorta because of the relatively large number of substitutions that would have been necessary. Altogether only 10 mean values were inserted accounting for <1% of the total data.

Results

The examination was generally completed without difficulty except for the recording of velocities from the abdominal aorta. It was possible to record velocities from this site in only 89 out of 110 subjects, and the angle in the visualised planes was often as large as 45° and at times even greater. Velocity recording also failed for the pulmonary artery in two subjects, for the ascending aorta in three, and for the descending aorta in five. Figure 1 shows examples of typical normal waveforms.

A normal distribution of modal peak velocity and time to peak velocity was observed at all sites. Table 1 shows the range of velocity at each site and the time to peak velocity (acceleration time) in the ascending aorta, the descending aorta, and the pulmonary artery. Figures 2–5 and Table 2 show the correlation

Table 2 *Correlation of modal peak velocities and time to peak velocity with body surface area and age*

Site	*Body surface area*			*Age*		
	r	*SEE*	*p value*	*r*	*SEE*	*p value*
Superior vena cava	−0·53	10·8	<0·001	−0·48	11·1	<0·001
Tricuspid valve	−0·22	12·2	<0·05	−0·25	12·1	<0·01
Pulmonary artery	−0·21	16·8	<0·05	−0·31	16·4	<0·01
Mitral valve	−0·27	15·3	<0·01	−0·39	14·7	<0·001
Ascending aorta	0·04	18·6	NS	−0·06	18·6	NS
Descending aorta	−0·07	16·9	NS	−0·04	16·9	NS
Time to peak velocity:						
Pulmonary artery	0·6	21·6	<0·001	0·61	21·4	<0·001
Ascending aorta	0·62	17·7	<0·001	0·53	19·2	<0·001
Descending aorta	0·57	23·3	<0·001	0·55	23·8	<0·001

SEE, standard error of estimate.

Table 3 *Correlation of modal peak velocity and time to peak velocity at different sites*

Site	*Modal peak velocity*						*Time to peak velocity*	
	SVC	*TV*	*PV*	*MV*	*Ascending aorta*	*Descending aorta*	*PV*	*Ascending aorta*
Modal peak velocity:								
Tricuspid valve (TV)	0·18							
Pulmonary valve (PV)	0·33	0·28						
Mitral valve (MV)	0·21	0·30	0·26					
Ascending aorta	0·10	0·07	0·47	0·29				
Descending aorta	0·14	0·06	0·40	0·32	0·50			
Time to peak velocity:								
Pulmonary valve	−0·39	−0·19	−0·25	−0·10	0·06	0·04		
Ascending aorta	−0·38	−0·05	−0·19	−0·34	−0·08	−0·07	0·45	
Descending aorta	−0·30	−0·13	−0·17	−0·16	0·14	0·01	0·54	0·59

SVC, superior vena cava.

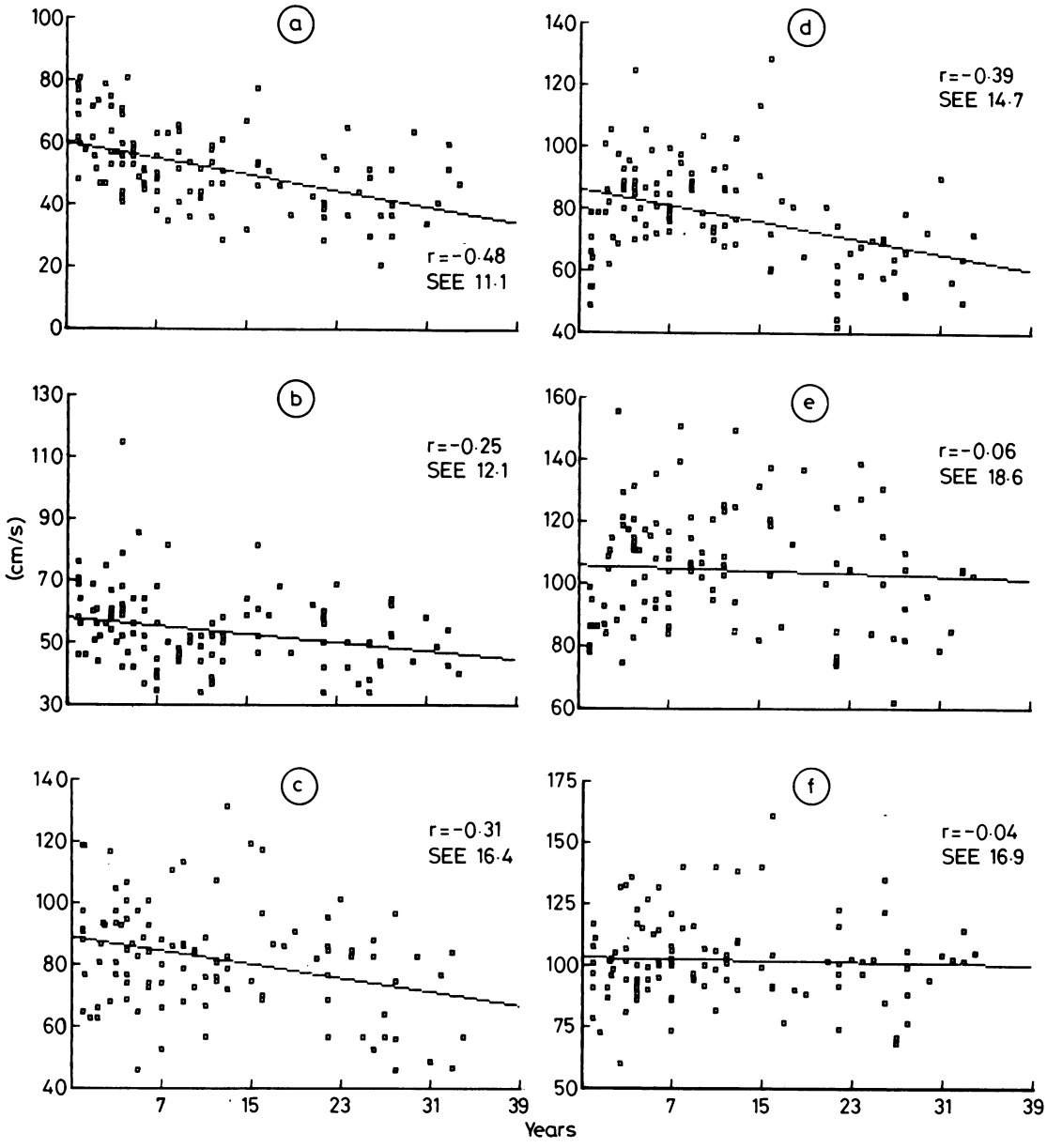

Fig. 2 *Correlation of age (years) with modal peak velocity (cm/s) (a) in the superior vena cava, (b) across the tricuspid valve, (c) in the pulmonary artery, (d) across the mitral valve, (e) in the ascending aorta, and (f) in the descending aorta.*

of the measured variables with age and body surface area. At most sites a statistically significant inverse relation between modal peak velocity and both age and body surface area was found. In the ascending and descending aorta, however, the correlation coefficients with age and body surface area were close to zero.

For time to peak velocity in the pulmonary artery and in the ascending and descending aorta there was a strong positive correlation with both age and body

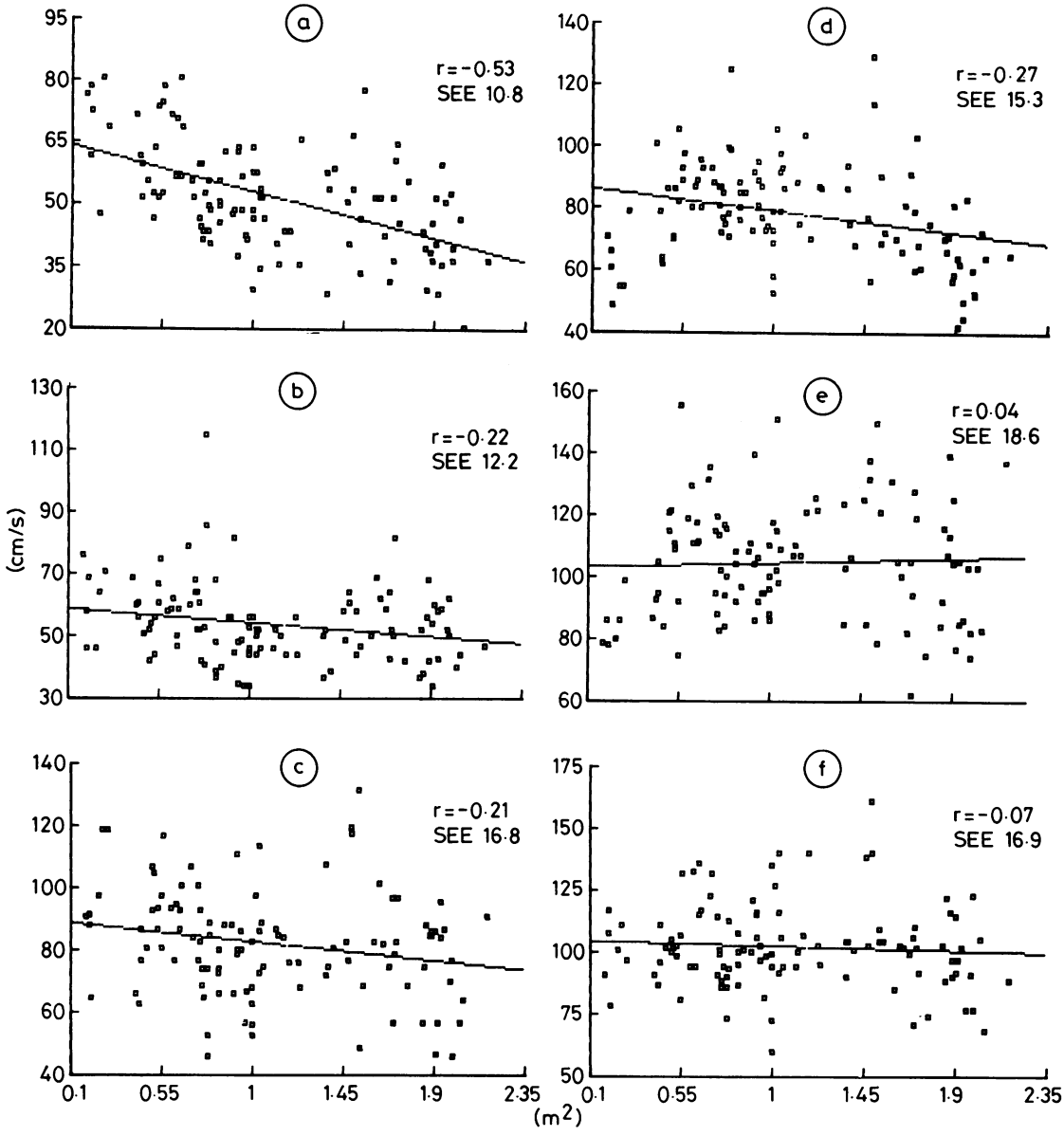

Fig. 3 *Correlation of body surface area (m^2) with modal peak velocity (cm/s) (a) in the superior vena cava, (b) across the tricuspid valve, (c) in the pulmonary artery, (d) across the mitral valve, (e) in the ascending aorta, and (f) in the descending aorta.*

surface area. Modal peak velocity generally did not correlate strongly at different sites (Table 3). The correlation of peak velocity in the ascending aorta with that in the descending aorta (Fig. 5) was, however, 0·5 ($p<0·001$).

Discussion

Doppler echocardiography is a useful tool in the investigation of patients with congenital cardiac disease since it allows blood velocity at various cardiac

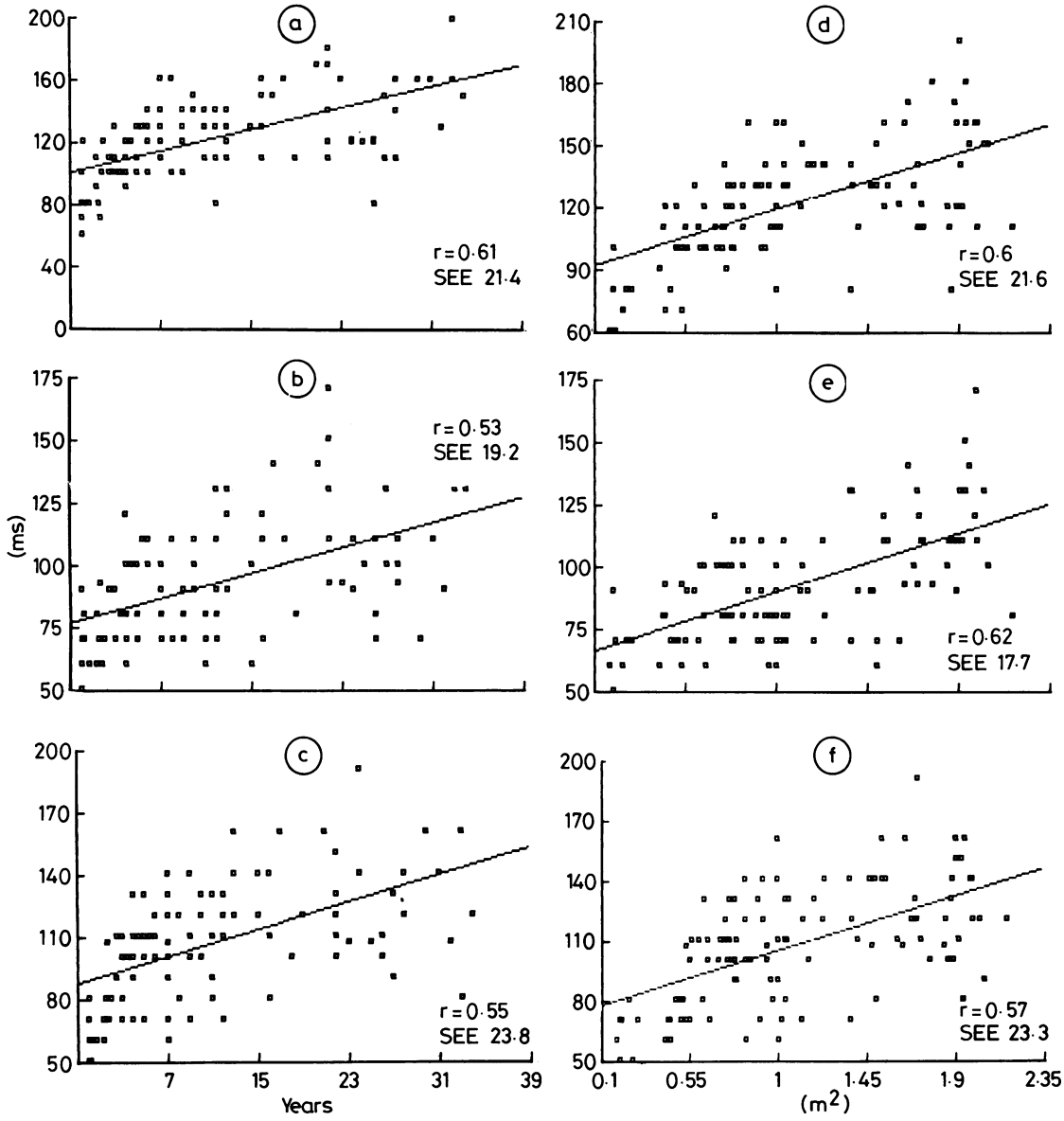

Fig. 4 *Correlation of age (years) and body surface area (m^2) with time to peak velocity (ms) ((a) and (d)) in the pulmonary artery, ((b) and (e)) in the ascending aorta, and ((c) and (f)) in the descending aorta.*

and vascular sites to be measured non-invasively. Velocity measurement, combined with other measurements, permits a quantitative assessment of blood flow and pressure gradients.[1-3] A detailed knowledge of normal values at clinically useful sites is clearly a prerequisite for interpreting data from patients with abnormal anatomy or haemodynamics.

Previous studies, mainly of adult populations, have reported normal values from a limited number of sites,[4-6] some using the continuous wave ultrasound

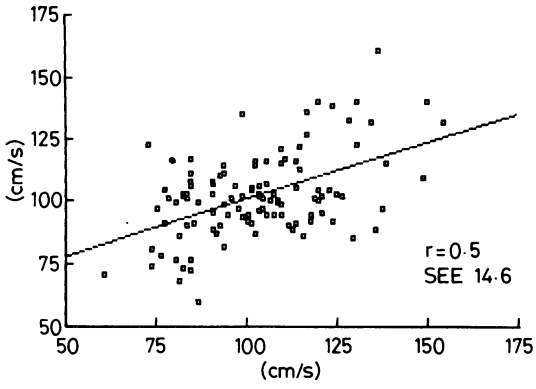

Fig. 5 *Correlation of modal peak velocity (cm/s) in the ascending aorta (vertical axis) with that in the descending aorta (horizontal axis).*

technique. In an earlier study of children by Grenadier and colleagues alignment of the ultrasound beam with blood flow in the third (elevational) plane was not confirmed[5]; thus the reported velocities may have underestimated true velocity. Our results confirm this for some sites.

PROBLEM OF ANGLE CORRECTION

Velocity (V) is calculated from the formula: V=(C dF)/(2F cos theta) where F is the emitted signal frequency, C the velocity of ultrasound in soft tissue, dF the frequency shift, and theta the intercept angle of the ultrasound beam with the direction of blood flow. Accurate measurement of the intercept angle in three planes is impossible with current Doppler instruments. If, however, theta is $<20°$ cosine theta is $\geq 0{\cdot}94$ and the recorded velocity is correct to within 6% of true velocity. Alignment of the ultrasound beam with flow by the method described should therefore permit a reasonably accurate measurement of maximum velocity without using a potentially inaccurate correction for the intercept angle.

USE OF MODAL PEAK VELOCITY

By definition the modal velocity is that velocity at which most of the blood cells within the sample volume are travelling at any moment in time. On the spectral display it appears as the densest area of the waveform (Fig. 1). During the acceleration phase, virtually all the cells in the sample volume are moving in the same direction at the same velocity and the waveform is a narrow line. At peak velocity there is some broadening of the waveform, although with accurate alignment with blood flow this rarely exceeds a velocity range of >25 cm/s. The peak velocity at the outer edge of the Doppler waveform is a point which, in practice, we have found more difficult to identify precisely, and in our experience modal peak velocity is a more reproducible point from which to make measurements. Although range adjustment will broaden or narrow the spectral display, the value of peak modal velocity will be unchanged.

With the exception of the abdominal aorta, imaged from the subcostal position, the methods we used allowed good alignment with flow at all sites. Modal peak velocity in the abdominal aorta had the widest range and highest standard deviation of any measurement in this study. This variability and the poor correlation between descending and abdominal aortic velocities probably resulted from an inadequate estimate of the spatial intercept angle between the ultrasound beam and blood flow in the abdominal aorta rather than any true difference in modal peak velocity between these two sites. Since the large intercept angle was necessary even small errors in estimation of this angle could make a considerable difference in the calculated velocity. The results suggest that using this method a difference in velocity between these two points could not be regarded as satisfactory evidence of aortic coarctation.

Although at most sites the modal peak velocities recorded in this study are similar to previously reported values[4-6] some points of difference emerge. Not all authors have, however, specified the point from which the measurements were made, and some of these differences may be due to differences in measurement technique. Our mean values for modal peak velocity are close to those of Grenadier *et al*[5] measured using the same method, but the range of values—particularly in the pulmonary artery and at the mitral inlet—are much wider in the present study. Some individuals attained modal peak velocities of approximately 130 cm/s at these sites compared with values of 90–105 cm/s in the study of Grenadier. At these two sites our highest values are more in accord with those of Hatle and Angelsen recorded using a combination of the pulsed and continuous wave Doppler techniques.[3] Nevertheless, Hatle and Angelsen[3] recorded maximal velocities in the aorta far in excess of ours for reasons which we cannot explain. At this site our values are much closer to those of Mowat *et al*.[6]

Within the age range of the subjects in this study there was no change in aortic modal peak velocity with increasing age (Fig. 2c), a finding in keeping with the observations of Light in 120 children aged from 1 to 12 years.[7] In a study of an older population maximal aortic velocity was found to decline with age.[6]

Mahan *et al* found an inverse relation between pulmonary artery acceleration time (time to peak velocity) and pulmonary artery pressure measured at

cardiac catheterisation.[8] In the present study pulmonary artery acceleration time is shown to be directly related to age (Fig. 4a), the lowest value of 55 ms occurring in the youngest subject aged 2 weeks. In view of the findings of Mahan *et al*[8] this short time to peak velocity might be attributed to the persistence of a relatively high pulmonary vasomotor tone in these youngest subjects, with some increase in pulmonary artery pressure. No observations are, however, available to confirm this, and at present acceleration times in this age group should be interpreted with caution.

Measurement of modal peak velocity was easy for the ascending aorta but more difficult for the descending aorta. The best alignment with flow is achieved at the junction of the transverse and descending aorta. Nevertheless, an appreciable degree of spectral broadening occurs in this location because cells within the sample volume are changing direction. None the less, ranges for ascending and descending aortic velocity are similar. Furthermore, our results show little velocity change at either site over the age range studied. Time to peak velocity in the ascending and descending aorta lengthened with age.

In accord with previous findings[4 5] modal peak velocity across the mitral valve exceeded that across the tricuspid valve (mean 53 (range 33–81) cm/s for the tricuspid and mean 81 (range 44–128) cm/s for the mitral valve). Superior vena caval velocity varied considerably with respiratory excursion and with increased intra-abdominal tension such as occurs with crying or upper respiratory tract obstruction. In this study all superior vena caval measurements were made with the subject at rest and breathing quietly. If any conclusion is to be drawn from a superior vena caval velocity pattern the above factors must be taken into account. An example of the use of normal superior vana caval velocities occurred during our study when we had the opportunity to examine two infants with total anomalous pulmonary venous connexion of the supracardiac type in whom the ascending pulmonary vein connected to the innominate vein. In these children superior vena caval blood velocity was increased at rest.

References

1 Huntsman LL, Stewart DK, Barnes SR, Franklin SB, Colocousis JS, Hessel EA. Noninvasive Doppler determination of cardiac output in man: clinical validation. *Circulation* 1983; **67**: 593–602.

2 Alverson DC, Eldridge M, Dillon T, Yabek SM, Berman W Jr. Noninvasive pulsed Doppler determination of cardiac output in neonates and children. *J Pediatr* 1982; **101**: 46–51.

3 Hatle L, Angelsen BA, Tromsdal A. Non-invasive assessment of aortic stenosis by Doppler ultrasound. *Br Heart J* 1980; **43**: 284–92.

4 Hatle L, Angelsen B. *Doppler ultrasound in cardiology: physical principles and clinical applications*. Philadelphia: Lea and Febiger, 1982: 72.

5 Grenadier E, Oliveira Lima C, Allen HD, *et al*. Normal intracardiac and great vessel Doppler flow velocities in infants and children. *J Am Coll Cardiol* 1980; **4**: 343–50.

6 Mowat DHR, Haites NE, Rawles JM. Aortic blood velocity measurement in healthy adults using a simple ultrasound technique. *Cardiovasc Res* 1983; **17**: 75–80.

7 Light LH. Implications of aortic blood velocity measurements in children [Abstract]. *J Physiol* 1978; **285**: 17P–18P.

8 Mahan G, Dabestani A, Gardin J, Allfie A, Burn C, Henry W. Estimate of pulmonary artery pressure by pulsed Doppler echocardiography [Abstract]. *Circulation* 1983; **68** (suppl III): 367.